

This tremor in intended movements lessens as the disease advances, and finally gives place to peculiar spasmodic contractions, especially of the extensor muscles, by which marked abnormalities of the joints are occasioned.

In hypochondriacal paralysis the subjective troubles are much more marked than those which are demonstrable as objective. Moreover, the paralyzes are only slight; they change about and rapidly disappear. Aggravations are occasionally induced by dyspeptic disturbances. The basis of these difficulties is a venous hyperæmia of the lower part of the cord.

Patients suffering from the anæmic forms of spinal irritation are for the most part pessimists. True, paralyzes are for the most part absent. There is only functional weakness, often caused by previous losses of semen to great extent. Pains occur in changing places; the bladder and rectum remain unaffected.

The general condition in real diseases of the cord remains often enough perfectly good for years, while the opposite is the case in spinal irritation. —*Deutsche Zeitschrift f. prakt. Medizin*, Nov. 23, 1878 (*Lancet and Clinico*).

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LOCOMOTOR ATAXIA.—Dr. A. Takacs publishes in the *Centralbl.* (50, 1878) the following clinical and anatomical results of his investigations on locomotor ataxia, promising a speedy publication of the details and methods.

1. The grey degeneration of the posterior columns, existing almost always in tabes, is a secondary process. The primary affection is an atrophy of the posterior roots, or the posterior cornua, or a posterior meningitis.

2. The constant anomaly of sensibility in tabes is a diminished velocity of conduction. Anæsthesia, however, or hyperæsthesia, do not occur in all cases.

3. The posterior columns contain centripetal fibres, nerves of touch. The grey substance conducts in the normal state only painful impressions (Schiff), but in morbid changes of the posterior columns it can assume the function of the posterior columns (Friedreich). This accounts for the diminished conductivity.

4. In a normal movement the muscles do not contract suddenly, but in variable phases, according to the intended movement. For this purpose the muscles require a graded stimulation, originating in the sensitive nerves, which perceive the muscle contraction.

5. But if this stimulation, starting in the sensory nerves, is retarded, the muscular contraction becomes also retarded, and hence jerking—*ataxic*.

6. In tabes the retardation of sensitive conduction is due to the fact, that the slowly conducting grey substance is fulfilling the function of the degenerated posterior columns.

7. When the posterior columns alone are degenerated, only ataxia occurs; but if the degeneration involves also the posterior roots and cornua, there will be found anæsthesia, or hyperæsthesia, according to the stage of the disease.

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CONTRACTURES IN INTRA-VENTRICULAR EFFUSIONS.—At the session of the Société de Biologie, March 1 (rep. in *Le Progrès Médical*), M. Cossy related

some experiments in which he had injected liquids directly into the ventricles of the brain. M. Duret had accounted for the contractures observed in intra-ventricular hemorrhages, and in experimental injections of the ventricles, by presuming an excitation acting on the parts forming the floor of the fourth ventricle, especially the restiform bodies. M. Cossy rejected this explanation, since in his experiments he had found the coagulated liquid injected only in the lateral and middle ventricles, and yet the contracture had been produced. He therefore concluded that it must be due to the irritation of the subjacent excito-motor parts (internal capsule and cerebral peduncles) by the sudden compression of the walls of the lateral and middle ventricles.

In like manner he explains the contracture in cases of hemorrhage limited to the lateral ventricles; not admitting the explanation of Duret that it is caused by the shock transmitted through the cephalo-rachidian fluid to the floor of the fourth ventricle. He remarked that convulsions were not any more frequent in cases where the effusion had reached the fourth than in those in which it remained limited to the lateral ventricles. Out of twenty cases of ventricular hemorrhage in which the blood reached the fourth ventricle, in eight only were there convulsions, in twelve they were lacking.

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HYDROPHOBIA.—T. B. Curtis, *Boston Medical and Surgical Journal*, Nov. 7 and 14, 1878, gives an account of a case of hydrophobia very well observed, and follows it with a discussion of the pathology of the disease. After passing in review the various causes of the paroxysms and their *facies*, he expresses the opinion that there are no involuntary convulsions whatever in hydrophobia, but that the spasms "consist in sudden attacks of *paralytic apnœa*, due to temporary, partial or complete, inhibition of the respiratory centre, taking place under the influence of peripheral impressions. The inhibitory stimulus proceeds first from the area of distribution of the superior laryngeal nerve, being originated by attempts to drink and by accumulated saliva; secondly, from the area of the fifth pair, as a result of wetting the lips or face, and of fanning; thirdly, from the nerves of sensation of the trunk and limbs; fourthly, from the nerves of special sense; and fifthly, from the emotional regions of the hemispheres; a paroxysm being excited by the fear of a paroxysm." Thus the characteristic phenomena of hydrophobia consist in merely the exaggeration of a normal physiological process—that of respiratory arrest or inhibition.

That it is a diminished resistance of the respiratory centre, and not an increased stimulus that is at the bottom of these effects, he thinks is indicated by the general permanent diminution of respiratory activity observed in these cases of hydrophobia, which is due to actual structural changes in the medulla. This is aided in its morbid action by additional depressing influence from the emotional cortical regions.

The following is therefore his scheme of the pathology of the disease:

"The activity of the respiratory centre—being kept in a permanent state of depression, 'below par,' so to speak, in consequence of local alterations of structure, and also in consequence of depressing influences proceeding from the diseased emotional regions of the brain—is thereby rendered liable